

Figure Legend

Supplemental Figure S1. Role of p38 MAPK, AMPK, and ERK in mediating the effect of FGF-19 insulin-induced expression of lipogenic enzymes. (A) Effect of FGF-19 on the phosphorylation of p38MAPK, AMPK and ERK1/2. Primary hepatocyte cultures were prepared and incubated in Waymouth's medium containing insulin. At 36 h of incubation, FGF-19 (50 ng/ml) was added to the culture medium and the incubation was continued for 10 min, 1 h, 6 h, and 12 h. Cells were harvested, total cell extracts were prepared, and Western analyses were performed using antibodies against phosphorylated p38MAPK (Thr¹⁸⁰/Tyr¹⁸²), phosphorylated AMPK (Thr¹⁷²), phosphorylated ERK1/2 (Thr²⁰²/Tyr²⁰⁴), total p38MAPK, total AMPK, and total ERK1/2. The data are representative of four independent experiments. (B) Effect of inhibiting ERK1/2 activity on FGF-19 regulation of GK and SREBP-1c expression. Primary hepatocyte cultures were prepared and incubated in Waymouth's medium containing insulin. At 35 h of incubation, U0126 (25 mM) or DMSO (vehicle) was added to the culture medium. FGF-19 was added to the culture medium at 36 h of incubation and the incubation was continued for 6 h. Cells were harvested and total RNA was isolated. GK mRNA and SREBP-1c mRNA were measured by quantitative real-time PCR. The level of mRNA in cells incubated with insulin and vehicle was set at 1, and the other values were adjusted proportionately. The percentage inhibition by FGF-19 was calculated for cells treated with vehicle and U0126. Values were calculated for individual experiments and then averaged. Values are means \pm four experiments. (C) The abundance of phosphorylated ERK1/2 and total ERK1/2 in total cell lysates was measured by Western analysis.

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